Chapter 8

SMOKERS AND NONSMOKERS OUTDOORS

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The level of air pollution is an important consideration in evaluating the significance of carboxyhemoglobin in the blood of smokers and nonsmokers. The carbon monoxide in the ambient air from moving or vehicular sources, as well as from stationary or occupational emission, is absorbed in the blood as readily as that contained in cigarette smoke. The amount detected in blood as carboxyhemoglobin represents the sum of all the exogenous sources plus the amount produced endogenously in the body.

It has been suggested that low levels of carboxyhemoglobin (about 5 to 10% saturation) are fatal to patients with heart or bronchopulmonary disease. The suspicion is based on the interference by carboxyhemoglobin with the oxygen-carrying capacity of the blood. It is further postulated that the diseased heart would suffer from lack of oxygen and that the lungs will not deliver sufficient amounts of oxygen to the alveolar capillaries for absorption by the blood with elevated carboxyhemoglobin. The result is myocardial infarction and death in the cardiac patient, and dyspnea and respiratory failure in the patient with bronchopulmonary disease.

The above hypothesis on the dangers of low level of carboxyhemoglobin is based on interpolation of results of several epidemiologic and experimental observations that are reviewed in this chapter. The problem is largely of interpretation of data and their relevance to both the cigarette smoker and the nonsmoker with preexisting cardiopulmonary disease. The question of smoking per se in the causation of disease has been discussed in Chapters 2, 3, and 4.

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A. Patients with Cardiac Disease

In the June, 1976 issue of Annal of Internal Medicine, Green, Cohen and Kurland[1] reported a 44-year old male who collapsed on the streets of Boston on a day when ambient temperature was 26°C and humidity was high.

If the man lived a sedentary style and was a heavy smoker, he would be considered as one example of sudden deaths from acute myocardial infarction with arteriosclerotic coronaries, that have been reported repeatedly in the literature during the past 20 years. The amount of air pollution, together with carboxyhemoglobin from smoking would be said to have triggered the fatal event.

But the patient was a marathon runner who was a nonsmoker; death was from myocardial infarction but the coronaries were "widely patent" and there was no evidence of old or recurrent occlusion.

Green et al.[1] reported the case to be the first documentation of a myocardial infarction in a trained athlete while participating in a marathon.

Advocates of long distance running for prevention of or rehabilitation from ischemic heart disease should be not only aware of this possible complication, but also should question the supposition that episodic maximal exertion bestows a beneficial effect on the coronary arteries. How adequate is the exercise performance test for evaluating a beneficial effect on the coronary arteries? Since the exercise test has wrongly led to misconceptions on the value of marathon running, can the test continue to be accepted in the evaluation of the effects of air pollutants?

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1. Exercise Performance Test.

The results of Aronow et al. on anginal patients on a treadmill have been reviewed in Chapter 2. The reduction in exercise time until the onset of angina was reduced in patients who have been exposed to freeway atmosphere or to inhalation of low level carbon monoxide. Although the treadmill showed a reduction in performance in subjects inhaling carbon monoxide,[2-9] actual athletic performances in competitive meets of cross-country track runners is not effected by carbon monoxide concentration but is correlated with the oxidant level in the ambient air.[10]

2. Mortality Statistics.

In an analysis of 1,075 coroners' cases in Los Angeles, Curphrey et al.[11] found no correlation between the incidence of myocardial infarction with carboxyhemoglobin levels, for both nonsmokers as well as smokers. Goldsmith et al.[12,13] arrived at an opposite conclusion, i.e. fatality rate among 3,080 patients with myocardial infarction admitted to 34 Los Angeles hospitals is greater in high pollution areas than in low pollution areas. The difference in conclusion is based on the source, i.e. hospital records against coroners' cases. It is pertinent to point out that a similar study conducted in Baltimore failed to find a correlation between carboxyhemoglobin level and incidence of heart attacks. (Chapter 3). Consideration of other related information indicates that the incidence of myocardial infarction is not correlated with the level of carbon monoxide exposure.

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B. Patients with Bronchopulmonary Disease.

Although the incidence of respiratory symptoms relates to the level of carboxyhemoglobin in the blood,[14] the coincidental exposure to air pollutants is the determining factor.[15, 16] This suspicion is based on the experimental work showing that carbon monoxide does not induce dyspnea or produce lung lesions, whereas pollutants such as sulfur dioxide, nitrogen oxides and hydrocarbons do (Chapter 7). Reichel et al.[17] exposed 44 persons to street traffic for three hours and found no respiratory restrictions indicating that the effect is seen after prolonged exposure to pollutants.

The correlation of levels of air pollution with incidence of respiratory disease was detected by Sterling et al.[18, 19] in an analysis of morbidity as indicated by the length of stay in Los Angeles hospitals of patients. Smoking habits were not included in the analysis so that it is not possible to verify the earlier conclusion of Goldsmith and Deane[20, 21] that respiratory symptoms were found to occur more frequently among cigarette smokers than nonsmokers of outside workman in the Los Angeles area.

C. Carboxyhemoglobin Levels Following Exposure to Atmospheric Pollution

1. Blood Samples Before and After Work.

The uptake of carbon monoxide from the ambient air has been demonstrated

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by analysis of blood samples from traffic policemen, pedestrians and commuters. A rise in carboxyhemoglobin values reflects the amount of carbon monoxide absorbed. In some studies, a distinction is made between smokers and nonsmokers. The results obtained by De Bruin et al.[22-24] from a group of policemen in Amsterdam are as follows:

Subjects (no.)	Before Work Mean ± S.D.	After Work Mean ± S.D.	Δ Mean
smokers (14)	4.62 ± 0.97	5.16 ± 0.81	+0.54
nonsmokers (10)	1.43 ± 0.26	1.66 ± 0.27	+0.23

The uptake of carbon monoxide is small and is an indication of the level of pollution in the city of Amsterdam. In the center of Cairo, Emara et al.[25] reported the following mean values:

Subjects	Before (mean)	After (mean)	Δ (mean)
smokers	1.9	9.5	7.6
nonsmokers	0.9	4.5	3.6

There is no mention of range of values and the number of subjects so that it is impossible to interpret the increased uptake of smokers compared to nonsmokers.

Morando and Rovida[26] reported opposite effects in traffic policemen during the course of their work at Via Milano, Genova.

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Subjects	Before (mean)	After (mean)	Δ (mean)
smokers	3.40	3.00	-0.40
nonsmokers	1.83	2.20	+0.37

The reduction in carboxyhemoglobin levels among smokers is in contrast to the increase in nonsmokers. The results are different from those reported from Cairo and Amsterdam.

2. Single Blood Samples.

The remaining studies of carboxyhemoglobin levels consist of single blood samples per individual, taken before, during or after exposure to the polluted air. The differences between smokers and nonsmokers are as follows:

Subjects	Smokers % COHb Mean ± S.D. (Range)	Nonsmokers % COHb Mean ± S.D. (Range)
New York City		
Bridge operators[27]	5.01 ± 2.25	2.93 ± 1.36
Pedestrians[27]	3.84 ± 2.06	1.94 ± 0.62
Stockholm		
Policemen[28]	3.5 ± 1.17 (1.5-6.2)	1.2 ± 1.17 (0.5-2.0)
Malmo		
Policemen[28]	5.0 ±2.44 (3.2-9.7)	0.8 ± 0.14 (0.7-1.1)
Erlangen - Nurnberg		
Dustmen[29]	5.1 ± 2.7	1.4 ± 0.64

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	Smokers % COHb	Nonsmokers % COHb
Athens		
Pedestrians[30]	(2-4)	(0.5-1.0)
Bordeaux		
Policemen[31]	2.35	1.23
Milan		
Pedestrians[32]	2.65 ± 0.99	1.60 ± 0.64

The last study tabulated above is by Grisler et al.[32] who reported differences in subjects living in the center of the city compound from those observed among rural inhabitants.

The above results indicate considerable overlapping in values for smokers and nonsmokers. The same phenomenon has been discussed in earlier chapters.

D. Practical Aspects of Carbon Monoxide in the Ambient Air

Smokers and nonsmokers respond differently in the amount of carbon monoxide they absorbed from the ambient air. The difference is not fully appreciated because the results of various studies show that the uptake of carbon monoxide is either higher or lower in smokers than in nonsmokers. The explanations for the variability are discussed in Chapter 1 and largely depend on the preexisting level of carboxyhemoglobin for both groups.

The information reviewed in this chapter has been used by organizations as bases for recommended air quality standards.[33-36] The amount of carbon

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monoxide from the ambient air outdoors is usually overlooked in the consideration of carboxyhemoglobin levels of smokers and nonsmokers.

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